interprets the higher odds ratio of 3.24 for women smoking 10 to 19 cigarettes per day compared with nonsmokers (in the National Institute of Child Health [NICHD] SIDS Cooperative Epidemiological Study Control A group) to be significantly greater than the odds ratio of 2.49 among women smoking fewer than 10 cigarettes per day, compared with nonsmokers. Somehow Dr Golding missed seeing the confidence intervals for these odds ratios (in Table 1 of the paper), for, if she had, she would have appreciated the considerable overlap of confidence intervals, which suggests no significant dose-response effect of smoking for the NICHD SIDS Cooperative Study. Thus, we do not find a discrepancy between our analysis and our conclusions.

Dr Golding also assumes that because data on smoking was gathered prior to the sudden infant death syndrome event in the Missouri population, it should be more valid than the smoking information gathered in the NICHD SIDS Cooperative Epidemiological Study (a case-control study). As we state in the paper, we have no way of knowing the direction that recall bias would take across the various categories of smoking if recall bias was present in the NICHD SIDS Cooperative Study. However, Drews and colleagues have investigated the issue of recall bias on 25 study variables (maternal and infant medical data, but not including smoking) in the NICHD study. They summarized their study by stating that "case-control differences in recall accuracy did not appear to create spurious associations with SIDS or to bias most associations away from the null value."[2] Recently, Gibbons and coworkers have examined the issue of recall bias in sudden infant death syndrome studies in which prospective and retrospective responses were obtained to an identical set of questions (including smoking and number of cigarettes smoked) asked of sudden infant death syndrome and control mothers.[3] They found a high level of agreement between case and control mothers for most variables, including the smoking variables. Thus, we are not willing to attribute greater validity to the data from the Missouri population over that of the NICHD SIDS Epidemiological Study population. □

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Chlorination or Ozonation?

In their excellent meta-analysis of water chlorination and cancer, Morris et al.[1] summarized the many problems encountered in assessing cumulative exposure to an environmental risk factor. Their description of the potentially harmful byproducts formed during chlorination was succinct. The statistical analysis was well handled, and the separate meta-analysis of those studies that controlled for selected confounders addressed appropriately the problems of confounding.

Of course, confounding by factors not considered in the original studies cannot be adjusted for and should be addressed in future prospective studies of this issue. For instance, the authors' conclusion that colon cancer is not associated with chlorination is tentative because of the nature of the studies available for their meta-analysis. Nonetheless, they have presented the best evidence for and against the associations researchers have found between chlorination and various cancers. The dose-response curve for the two cancer sites (bladder and rectal) significantly related to chlorination, together with (a) the consistent results from different data combinations and (b) the power estimates, is reassuring that this relationship will hold in future studies.

Tests for mutagenic and carcinogenic activity depend on many factors that are not always accounted for in longitudinal studies (e.g., type of organic matter, point at which oxidants are added, scale of study, etc.).[2] The situation becomes even less certain when considering low-dose exposures over many years (especially when combined with low-dose chronic exposures to other mutagenic chemicals from water and food). Because it raises serum cholesterol levels in animals,[3] chlorination may have other detrimental effects besides cancer. Chemicals such as the pesticide aldicarb are not removed by chlorination and may be associated with immunosuppression.[4]

Finally, the authors are correct in calling for implementation of "disinfection strategies that are not associated with adverse health effects." Los Angeles and other cities switched from chlorination to ozonation many years ago.[5,6] The US Environmental Protection Agency has been instrumental in establishing research guidelines and funding for disinfection alternatives.[6–9] One large-scale pilot study at a Louisiana water-treatment plant concluded that "ozone appears to be the disinfectant of choice because lower concentrations of organics were detected during its use."[6] The best combination may be ozonation with granulated activated charcoal (or sand) filtration, applied late in the process.[2,10,11] A reverse osmosis treatment "train" is an efficient alternative for home use.[12] □

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Morris Responds

Dr Chapdelaine raises several important points that my colleagues and I did not fully address in our original study.[1] Most of these issues relate to the limitations of available studies on the health effects of chlorine by-products. These limitations will certainly have an impact on the interpretation of the results of the meta-analysis.

First, the failure of the meta-analysis to find an association between chlorination by-products and colon cancer seems somewhat surprising in light of the by-products' significant association with rectal cancer. To some extent, this may reflect the lack of statistical power available to detect an effect. Table 1 lists results from a meta-analysis not presented in the original paper that evaluates the association between chlorination by-products and colon cancer by level of exposure. These results suggest that a dose-response relationship may exist for colon cancer, but, at any level of exposure, the risks are higher for rectal than colon cancer. This difference is biologically plausible. The rectum is exposed to excretory products at higher concentrations and for longer periods than the colon. At a given level of by-products in the tap water, the rectum is exposed to higher levels of potential carcinogens than is the colon.

Dr Chapdelaine also points out that the factors that control the quantity and nature of chlorination by-products in drinking water are not fully accounted for in the epidemiological studies conducted to date. Chlorination by-products include a broad range of compounds, many of which have not been characterized. The tendency for regulatory standards and quantitative exposure assessment to focus on trihalomethanes may be inappropriate. The demonstrated, potent mutagenicity of by-products produced at concentrations that are orders of magnitude lower than the levels for trihalomethanes[2] raises serious questions about this focus on trihalomethanes. It is likely that more than one of the many compounds produced during water chlorination may play a role in carcinogenesis, with the potential for synergistic effects. Furthermore, physiology and pharmacodynamics[3] would suggest that different routes of exposure and different by-products or sets of by-products may be responsible for carcinogenesis at different sites. For example, a carcinogen must cross more barriers and is more likely to be metabolized in some way if its target is the bladder as opposed to the rectum. Changes in the nature of the raw water supply and the precise conditions and methods for chlorination will have substantial influence on the nature of the by-products and the consequent, site-specific cancer risk.

Dr Chapdelaine expresses concern that cancer is not the only potential adverse outcome. As he points out, recent studies have suggested other detrimental effects from chlorination by-products. Teratogenic effects should be added to this list of potential health effects.[4]

Finally, Dr Chapdelaine suggests that ozonation should be pursued as an alternative to chlorination. Although the available data support the assertion that ozone is less likely than chlorine to produce carcinogens,[5] we should not be overzealous in rapidly shifting to other means of water disinfection. Most of the alternative methods of water disinfection (including ozonation) were available when chlorination of drinking water was first introduced in the early part of this century. Cost, ease of application, and, perhaps most important, the residual decontamination associated with chlorination rapidly made it the method of choice. Even with identified risks, the health benefits of water chlorination far exceed the known health risks. We should actively pursue alternatives to chlorination for water disinfection, but we must take care not to increase our risk of infectious diseases in the process. □

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Editor's Note. See related erratum (p 1257) in this issue.

TABLE 1—Meta-Analytic Estimates of the Association between Colorectal Cancer and Levels of Cumulative Exposure to Chlorination By-Products

Site	Level of Exposure	Relative Risk Estimate	95% Confidence Interval
Colon	Low	0.75	0.16, 3.54
	Moderate	1.06	0.60, 1.86
	High	1.20	0.79, 1.82
Rectum	Low	1.13	0.61, 2.09
	Moderate	1.29	1.00, 1.67
	High	2.04	1.18, 3.53

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What Does *America's Public Health Report Card* Reflect?

The project completed by the American Public Health Association (APHA), *America's Public Health Report Card,* has at least two main objectives: (a) to draw attention to the deteriorating status of public health across the United States and (b) to serve as a tool for the state-by-state assessment, planning, and evaluation of public health activities.[1] For the first objective the report card appears to have been an unequivocal success. With respect to the second, however, questions about methodology raise concerns about the usefulness of the document.

The report is light on methodological detail and collapses highly disparate variables to create rankings. The most notable result of this procedure is that meaningful